A Case Review of Intracranial Tuberculoma in an Immune-Competent Young Nigerian Woman

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Abstract: Intracranial tuberculomas are a rare but well-recognized complication of Tuberculosis. It is associated with high disease burden and mortality. The diagnosis of intracranial Tuberculoma remains a challenge in low-income countries and requires a high index of suspicion. We report a case of rare intracranial Tuberculoma in a young Nigerian woman. She presented with focal seizure, crawling sensation and rotatory movement of the left hand. She also had a history of cough and weight loss over three months. The main finding on clinical examination were features of cerebellar dysfunction on the left side. She had a brain CT scan, which showed multiple ring-enhancing hyperdense mass lesion in Right parietal lobe 8.6x6.6mm. The lesion had extensive peri-lesionaloedema with compression of the ipsilateral lateral ventricle, and a shift of parietal lobe to the Right of the midline. There is also an ill-defined isodense lesion in the left cerebellar hemisphere with peripheral ring enhancement on contrast administration. She had an elevated Erythrocyte sedimentation rate (ESR) and a negative HIV test. Her Gene-Xpert result was positive for mycobacterium tuberculosis. A diagnosis of intracranial Tuberculoma in an immune-competent woman was made, and the patient had treatment with anti-tuberculous medication. The patient had complete resolution of her clinical symptoms and a significant reduction in the size of the lesion on imaging studies. Intracranial Tuberculoma are rare conditions associated with significant morbidity and mortality. Diagnosis requires a high index of suspicion and for effective management.

Date of Submission: 16-03-2020 Date of Acceptance: 01-04-2020

I. Introduction

Tuberculosis is a multisystemic disease with a myriad of presentation and manifestation. It is the most common cause of infectious disease-related mortality worldwide. Tuberculosis primarily affects the lungs, extrapulmonary involvement of Tuberculosis do exist either in isolation or combined. Central Nervous System Tuberculosis (CNS-TB) represents 5-10% of extrapulmonary TB and accounts for approximately 1% of all TB cases.¹ Clinical forms of CNS TB include Tuberculous meningitis, Tuberculoma or tuberculous brain abscess. The incidence of CNS TB is related to the prevalence of TB in the community. It is the most common type of chronic central nervous system infection in developing countries. It is commoner among under five and immunocompromised adults.² It carries a high mortality and a distressing level of neurological morbidity. It remains a formidable diagnostic challenge because the burden of CNS TB lies in resource-starved regions of the world.

II. Case Report

We report a case of a 36-year-old married woman and wife of a long-distance driver. She presented with a cough of 3 months, left-sided weakness and vomiting of 2 months duration. The cough was of gradual onset productive of whitish sputum, with associated significant weight loss—no accompanying history of haemoptysis, low-grade fever or drenching night sweats. Left-sided weakness was noticed four weeks after the initial symptoms. The weakness was severe enough to prevent her from carrying out her house chores and holding small objects. There is associated focal stiffness and jerking of the Left hand. A crawling sensation usually precedes the jerking movement, uncontrollable circular movement of the Left palm, migrating to her shoulder, chest and subsequently feeling of impending passing out. The Left leg also feels like it is being moved laterally, weaker than other limbs: no dizziness, Vertigo, loss of consciousness. No history of auditory or visual disturbance.Vomiting was projectile in nature with occasional frontal headaches. The patient had 2-3 episodes per day—no abdominal pain, diarrhoea, constipation or change in bowel habit.

Examination reveals a chronically ill-looking woman, pale, with no significant lymphadenopathy. Her Right pupil: 3mm briskly reactive while the Left: 4mm sluggishly reactive to light. She had the following signs of Cerebellar dysfunction on the left side: intention tremor, Dysdiadokinesia, Impaired heel to shin test and

Tandem walk: sways to the left. She had left-sided myoclonic Jerks. Examination of the other systems was unremarkable.

Initial assessment of intracranial space-occupying lesion with background immunosuppressive disease (HIV/AIDS) was entertained.

On account of the above, she had a brain CT scan investigation, which showed multiple ring-enhancing hyperdense mass lesion in the Right parietal lobe with the largest measuring 8.6x6.6mm. The lesion had extensive peri-lesionaloedema, compression of the ipsilateral lateral ventricles and a shift of parietal lobe to the Right cerebral hemisphere. There is also an ill-defined isodense lesion in the left cerebellar hemisphere with peripheral ring enhancement on contrast administration. Patient brain computerized tomogram scan is in Figures1 and 2.



Figure 1

Figure 2

Figure 4

Chest radiograph showed military shadows in both lung field (Figure 4). Her retroviral screening result retuned negative for HIV. The erythrocyte sedimentation rate was high, 130mm/hour, on the Westergren stand. The Induced sputum Gene-Xpert result was positive for mycobacterium tuberculosis. The full blood count, electrolyte/ urea/ creatinine, and the liver function test was essentially normal

Overall diagnosis is Tuberculoma of the brain in an immunocompetent young adult. The patient commenced anti-tuberculosis drug, fixed-dose of 4 tablets combination of Isoniazid, Rifampicin, Pyrazinamide, Ethambutol (HRZE). Also, she received Tab pyridoxine 25m daily and Tab dexamethasone 4mg.

She made remarkable clinical improvement during the clinic visit. Weight appreciating, seizure subsided and complete resolution of her cough. She is still on the extension phase of anti-tuberculosis medication.

III. Discussion

Intracranial tuberculomas are a rare but well-recognized complication of TB. They are the least common presentation of CNS tuberculosis, accounting for 1% of these patients.¹ Tuberculomas are frequently encountered brain lesions in tropical countries. The diagnosis is made with a review of clinical presentation, epidemiologic, and imaging studies or via needle biopsy. In resource-limited countries, diagnosis is difficult, however a combination imaging studies and any surrogate evidence of mycobacterium tuberculosis infection may suffice.

The pathogenesis of CNS tuberculoma is similar to that of TB meningitis in the early stages. In contrast to TBM, the tubercles are walled-off from the brain parenchyma and meninges. This structure organizes into a complex lesion referred to as the Rich focus.³ Rupture of a Rich focus into the cerebrospinal fluid is thought to be the cause of tuberculous meningitis. They can also develop into a dense fibrous capsule. On gross examination of the lesions, they appear as well-circumscribed masses of varying sizes. A patient can have solitary or multiple lesions in only 15-34% of the cases, localized to any part of the brain.⁴ Tuberculomas can sometimes develop before or after treatment for TBM.

The patient under review had multiple intracranial space-occupying masses. The lesion was localized to the right parietal lobe and the left cerebellar hemisphere from the imaging studies. Besides the imaging evidence of ring-enhancing lesion in the brain, the patient had perilesional oedema with a mass effect shift of structure away from the midline. Important differential diagnosis of a ring-enhancing lesion in the brain includes; CNS lymphoma, CNS toxoplasmosis, pyogenic abscesses and metastatic brain tumours. In immune-compromised individuals CNS lymphoma, CNS toxoplasmosis is the first differential.⁵ whereas, in an immunocompetent host, tumours – both primary and metastatic – and pyogenic abscesses remain the most likely diagnosis.⁵Tumours on CT scan have thicker walls with more irregular margins. In comparison, Abscesses have a well-circumscribed thin and smoother outer margin.⁵ However, distinguishing these lesions of the brain based on CT or MRI alone is not adequate and comprehensive enough.

The clinical presentation of intracranial Tuberculoma is those of single or multiple intracranial spaceoccupying lesions, primarily seizures, Headache and other signs of raised intracranial pressure. The features of the seizure can be a pointer to the location of the Tuberculoma in the brain. Recognizing parietal lobe Seizures may be challenging, especially in children, because of the subjective nature of the experience that occurs. Seizures begin with contralateral (or rarely ipsilateral or bilateral) focal somatosensory seizures, most commonly paraesthesias with tingling and numbness. There may be prickling, tickling, crawling or electricshock sensations in the affected body part.⁶ The sensory abnormality may be localized at the onset but can spread subsequently along a body part as the seizure spreads on the cerebral cortex according to the sensory homunculus.⁶ The focal seizure with secondary spread may lead to motor activity in the affected body part. Commonly with rotatory body movements in the Ipsilateral or contralateral.⁶

The index case presented with somatosensory focal seizures on the contralateral side of the cerebral lesion with sequential spread on the cerebral cortex to affect the lower extremity. Also, the patient had clinical features of raised intracranial pressure. The lesion in the cerebellum accounted for the cerebellar dysfunction features seen during the clinical examination of the patients.

The clinical course of cerebral Tuberculoma presenting with seizures depends on the immune status of the patients. Studies have shown that cerebral Tuberculoma in immune-competent patients was more likely to present with seizure than immune-compromised individuals.⁷Intracranial Tuberculoma is often clinically quiescent for a long time, and a seizure can be an indicator of the presence of the disease in such cases. This pattern of presentation is rare and difficult to diagnose. Majority of cases, however, will present with accompanying features of systemic Tuberculosis. The final fate of the Tuberculoma will depend on the clinical course of the disease. If the caseous core of the Tuberculoma liquefies, a TB abscess results.⁸ These patients tend to be clinically worse overall than those with correspondingly sized Tuberculoma.⁸ Again, focal seizures can be an essential initial clinical manifestation.⁸ Alternatively, it will calcify.

The presence of cough, weight loss, elevated erythrocyte sedimentation rate, and reticulonodular lesion in the lung field of the chest radiograph and positive GeneXpert for all support the diagnosis of mycobacterium tuberculosis infection.

Concerning treatment, the patient received four tablets of HRZE for 6months and extended further for another 6months. The repeat MRI showed a significant reduction of the cerebral lesion and complete resolution of the cerebellar lesion (Figure 3). Sometimes paradoxical enlargement of the Tuberculoma during treatment can occur.⁹ Also, Two-thirds of patients still have image evidence of Tuberculoma after 12 months of standard therapy and complete resolution of the symptoms.¹⁰ The persistence of the ring-enhancing lesion could indicate an active lesion or inflammation. Systemic corticosteroids are kept specially for perilesional oedema, paradoxical progression during treatment, or when there is raised intracranial pressure.

IV. Conclusion

Intracranial Tuberculoma is a rare but occasional encountered medical condition in middle and low resource countries. Early diagnosis and prompt treatment are necessary to reduce the morbidity and mortality commonly associated with the condition.

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Akor Alexander Agada, etal. "A Case Review of Intracranial Tuberculoma in an Immune-Competent Young Nigerian Woman." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 19(3), 2020, pp. 05-08.

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